MARCH 1957 REPORT 149

Range Cattle Production, 7

GENETICS OF CATTLE

A Literature Review

By

C. B. ROUBICEK, R. T. CLARK and O. F. PAHNISH

A contribution from the W-1 Regional Research Project, "Improvement of Beef Cattle through the Application of Breeding Methods," in which the Western States — Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, Wyoming and the Territory of Hawaii — are cooperating with the Agricultural Research Service, United States Department of Agriculture.

Published by

Agricultural Experiment Station
University of Arizona
Tucson

CATTLE PRODUCTION

A Literature Review

Section VII

INHERITANCE

Carl B. Roubicek
Animal Science Department
University of Arizona

R. T. Clark
Coordinator, Beef Cattle Breeding Research
United States Department of Agriculture
Denver, Colorado

O. F. Pahnish
Animal Science Department
University of Arizona

TABLE OF CONTENTS

INHERITANCE

Chromosome Number

There is not complete agreement as to the chromosome number in cattle (134) (166). The diploid number has been reported by various investigators as 16, 20 to 25, 20 to 24, 33, 37, and 60 (68)(134)(136)(147). It would appear that the diploid number of 60 may be more nearly accurate. There is agreement that in cattle the male is XY and the female XX. The diploid number of the Indian water buffalo has been reported as 48 (45).

Inherited Factors Affecting Fertility

White Heifer Disease

The name is actually non-descriptive, although the condition is commonly found in white heifers of the Shorthorn breed. It has also been reported in roan and red Shorthorns and in colored animals of other breeds (18)(156). The condition appears to be due to failure of development of the Mullerian ducts, and is often referred to as "imperforate hymen" (156). Some of the more common characteristics are (64):

a. Closed hymen, or hymen persisting in varying degrees

b. Distention of one or both uterine horns, the uterine body being present in rudimentary form

c. Complete absence of cervix and anterior vagina

d. Prominence of Wolffian ducts

e. Presence of longitudinal submucous channels in the vagina

f. Aplasia of one uterine horn

This condition is apparently of genetic causation, but the exact mode of inheritance is not known.

Male Sterility, "Knobbed Spermatozoa"

Reported in Holstein-Friesian bulls, the condition is characterized by abnormal formation of the acrosome (40). Studies indicate that the formation of vacuoles in the developing spermheads may be related to some unknown changes in the nucleic acid metabolism of the spermhead. There is an absence of any chromosomal aberration, quantitative or structural. The condition does not appear to have a counterpart in the female, and since only males are affected it is concluded that the condition is due to an autosomal, sex-limited gene.

Sexual Gland Hypoplasia

The condition is reported in Swedish Highland cattle. It appears that the epithelium layers of the seminal ducts and follicles of the ovaries are underdeveloped. This may occur in either one or in both ovaries or testicles. Double-sided hypoplasia in females affects the secondary sexual characteristics. Double-sided hypoplasts in both sexes are sterile, although sexual instinct and copulation are normal. One-sided hypoplasts may have irregular reproduction ranging from very good to sterile (53).

The ovarian hypoplasia became a serious problem in the breed, affecting 17.5 percent of the cattle examined in 1936 (125). An all-out campaign to reduce the incidence was undertaken with financial assistance from the government. The incidence by 1948 had been reduced to 9.4 percent.

The condition was first introduced from one breeding center, and the incidence showed a progressive decrease with increasing distance from this breeding center (124).

The condition is due to a recessive autosomal gene with incomplete penetrance (123).

Atrophy of Testes

An examination of the testes of six sterile bulls showed gross and microscopic changes, including atrophy, calcification, degeneration of seminiferous tubules, and varying degrees of fibrosis (57). Mode of inheritance not determined.

Impotentia Coeundi

An inability in the male to copulate caused by failure of the sigmoid curve of the penis to straighten during coitus. Due to an autosomal recessive gene (64).

Umbilical Hernia

This condition may interfere with breeding efficiency. It has been reported as due to a sex-limited dominant gene of low penetrance (64), and also as due to one or more pairs of autosomal recessive factors (63).

Turned Tails

The sperm are abnormal with respect to their tails being turned back past the head. The mode of inheritance has not been determined (64).

Failure of Spermatogenesis

In crosses of European cattle with bison or yak, the inheritance seems to be for the large testicle of the European parent and the small scrotal sac from the other parent. This results in excessive temperature within the testicle and a consequent failure of spermatogenesis (64). This has been observed in crosses of (a) bison x domestic cattle, (b) yak x domestic cattle, and (c) yak x Zebu.

Endocrine Disorders

In male animals, a genetic predisposition toward inability to copulate and toward hormonal disturbances in spermatogenesis has been reported (123). No mode of inheritance is given.

Female Sterility

This general term is applied to a condition reported in Jersey and Holstein cows (75)(76). The cause of failure in the reproductive system has not been determined, but it is probably zygote abortion in the late cleavage or early blastocyst stage. The condition is due to a single autosomal recessive sex-limited factor (76)(117). Progeny tests have shown that sires may be homozygous for the female sterility gene (141).

Foetal Resorption

The fetus is found in various stages, from decomposed masses to only bones or dried mummies (46). The mode of inheritance is not known.

Mummification

Reported in Red Danish cattle, the fetus dies at the eighth month of gestation. The fetus shows a short neck, stiff legs, and prominent joints (46). Due to an autosomal recessive factor.

Cystic Ovaries

In Swedish Highland females, No exact mode of inheritance determined (123).

Intensity of Heat

Breed differences have been noted in degree of heat. Simmenthal, Telemark, and Swedish Highland cattle show intense heat. Swedish bred cattle show very weak heat and may be undetected.

Gonadless

The absence of an ovary has been shown to be due to an autosomal dominant (64).

Tubular Genitalia

The vulva and cervix are constricted. The mode of inheritance is not known (64).

Prolonged Gestation

Prolonged pregnancy in the Swedish Red-and-White cows has been reported from 332 to 510 days (86).

The calves all showed extreme hyperdevelopment of the body extremities, and in all cows the calves died. In the Holstein, gestation has been reported as 20 to 88 days overlong (74)(107). Calves weigh from 105 to 168 pounds, but are of normal proportions. It was concluded that the genotype of the fetus causes the prolonged gestation (74). In Japanese cattle, gestation periods of 311 to 403 days have occurred. It was suggested that the fetus is homozygous for a recessive gene. This may cause some secretory glands of the fetus to affect the maternal body conditions and disturb the onset of labor (106). The sire of the calf has been shown to have a significant influence on the gestation length in the Red Sindhi, Gir, and Tharparkar breeds in India (1).

Fertility of "Freemartin:"

Jersey cows, full sisters, born less than a year apart, each was a twin with a bull and each was fertile. Since only about 10 percent of the bisexed twin females are fertile, this occurrence suggests the possibility that the fertility of a heifer born twin to a male may be controlled by heredity (5).

Lethals and Sublethals

The division between lethal and sublethal is usually somewhat arbitrary and not too well defined. Under some conditions, or with certain modifying genes, a factor may behave as a true lethal, whereas a somewhat different environment or associated gene complex may cause the factor to behave in a much different way.

Dwarfism appears to be one of the characters in cattle that is present in many different forms.

Dwarfs in cattle have been recorded since 1860 (164). Dwarf types have been reported from many breeds, including (a) Aberdeen-Angus, (b) African Uganda (Zebu), (c) Ayrshire, (d) Dexter, (e) Guernsey, (f) Hereford, (g) Holstein-Friesian (American, British, Swedish), (h) Jaroslav, (i) Jersey, (j) Oplandske (Norway), (k) Shorthorn, (l) Swedish Red-and-White, (m) Spanish (Puerto Rico), (n) Telemark, (o) West African Shorthorn.

Dominant Achondroplasia

The "bulldog" calves of the Dexter breed in Ireland were described in detail in 1904. They have short, round heads; depressed nostrils; bulging foreheads; projected mandibles; flabby tongues; short vertebral columns; thick, loose skin; inguinal hernia, and short legs. They are about half normal size. They are aborted between the sixth and eighth month of the intra-uterine period. The dam has an accumulation of uterine fluid between the fourth and fifth month of gestation. Breeding tests showed that the condition is inherited as a single autosomal dominant (97).

The Dexter cattle are heterozygous. Inter se mating of Dexters resulted in segregations in the ratio of 1:2:1 (twenty-five percent of the offspring being homozygous normal Kerry type, fifty percent heterozygous Dexters, and twenty-five percent homozygous Dexter monsters). No bulldog calf was produced from the mating of Dexters with Kerry cattle.

"Duck-legged" cattle which appear to be similar in appearance to the Dexter breed have been reported in the United States (133). No dwarf or "bulldog" calves were found among the progeny of the "duck-legs". Some of the "duck-legged" cattle were confused with short-legged cattle of conventional breeding. The pituitaries of the "duck-legged" cattle were unusually small. The trait was due to a single dominant gene, although dominance is sometimes incomplete.

Dominant achondroplasia of the "bulldog" type also has been reported in the British Friesians (41) and in the Jersey (140).

Hereford breeders developed small-type cattle which were placing high in the show rings (190). They were short-legged and early maturing. These cattle were generally less extreme than the Dexter type, but were smaller at maturity than conventional Herefords. These small-type cattle apparently have a mildly hyperfunctioning thyroid compared with conventional-type steers (131).

"Compact" Shorthorns were described that resembled the small-type Herefords (172). They could be identified at birth and the characteristics were distinct throughout life. They appeared shorter of head, neck, body, and legs than the "standard" type of Shorthorn cattle. They appeared to be thicker in their muscles. Some of them had a tendency toward heavy shoulders and crooked legs. From field data available, it was concluded that the "compact" condition in Shorthorns is due to a single dominant gene.

Recessive Dwarfism

A recessive type of achondroplasia has been described which is referred to as the "Telemark type" (97). The heterozygous Telemark cattle appear normal. The monsters were born alive after full-term gestation and died within a few days from asphyxia (153). A known heterozygous Telemark bull was mated with eight Dexter cows. During the period 1926 to 1931, 24 calves and a mummy were produced.

These included five long-legged calves, eleven typical Dexter calves, and eight which were not classified--probably long-legged. No monster was produced. Neither the hypothesis of identical genes for both conditions nor the hypothesis of multiple allelomorphs could be supported because no monsters were produced in the above cross. The probable explanation was that these two lethals were quite independent, and that each breed carried the normal factor corresponding to the lethal of the other breed.

"Bulldog" calves have been reported in the African Uganda cattle (21). The Uganda breed of cattle is supposed to have originated from a cross of the long-horned Ankole breed and Easter Province animals which are typical Zebus of small size.

Three achondroplastic calves have been described in the Jaroslav breed in Russia (129). All three dwarfs resulted from the mating of champion cow "Zolataja" to her son, grandson, and half brother, respectively. The dwarfs had deformed lower jaws and shortened legs. One of these dwarfs could not eat grass and hay, but the other two, although they had shortened lower jaws, could eat normally. They did not live long. It was felt that this was a milder form of achondroplasia than other types which had been reported at that time. It was concluded that the sires were heterozygous for the monstrosity.

"Bulldog" calves were reported in the Guernsey, Jersey, and Ayrshire breeds, all apparently caused by the homozygous condition of a recessive gene (19). Dwarf cattle also are reported among the original Spanish cattle in Puerto Rico (4). A new sublethal type of achondroplasia was brought to light as a result of the inbreeding of Jersey cattle in California (71). It was recessive in nature, but more variable in its phenotypic expression than the Telemark type. The greatest modifications seemed to occur in the development of the bones of the skull and jaw. Defective calves could be identified at birth by a short, broad head, and a prominent forehead. Cleft palate might be observed in severe cases and the maxilla was sometimes shortened. The gene involved "has little, if any, effect on leg length." The dwarfs died after birth, although one achondroplastic female lived to 14 months of age.

Jersey "bulldog" calves that had notched ears have been described (178). In addition, they had shortened maxillae; bulging eyes; short, gnarled legs; no tails, and were hermaphrodites. They were full term and born alive. Jersey "bulldog" cattle are described as having skulls broader and shorter than normal (13). The nasal bone is particularly short and broad, and the poll is wider between the cores. The orbits are large and the upper jaw short. The animals apparently have impaired vision.

"Stumpy," a recessive achondroplasia in Shorthorns, has been described (10). This type of dwarfism was discovered following a line-breeding program in a pure-bred Shorthorn herd in central Nebraska. The dwarf or "stumpy" calves had three common characteristics: (a) They had curly coats and smaller switches. By these characteristics the "stumpy" individuals could be detected at birth, (b) An achondroplastic condition more marked in the forelegs than in the hindlegs. The knees were enlarged. The cannon bones were twisted. The body and head seemed to be normal in size, (c) Metabolic disturbances were present in all "stumpy" calves. Most of these animals were thin. These dwarfs lived and reproduced. Although the condition is not lethal, it is a serious economic loss to breeders. No post-mortem examinations were made and the endocrine glands were not studied. No measurements of the animals were taken. Between 1937 and 1946, a total of 562 calves were born in the inbreeding program. Among these were 26 dwarfs. The incidence of dwarfs in the herd for that period was, therefore, 4.6 percent.

All of the dwarfs had a common ancestor in which the mutation might have occurred. From an analysis of the pedigrees, it was concluded that the "stumpy" syndrome was caused by a single autosomal recessive gene.

West Africa Shorthorn cattle have been described that are dwarfed in stature, humpless, and do not resemble Zebu in any characteristics. A full-grown bull will weigh 350 pounds and measure about 36 inches to the top of the withers. They appear to breed true genetically since they do not change even under optimum environmental conditions (108).

An achondroplastic type found in Swedish Red-and-White cattle appears to be similar to dwarfs found in some of the British breeds (110):

a. Head short and broad with moderately bulging forehead

b. Upper jaw noticeably shorter than lower jaw

c. Legs comparatively short, particularly below knee and hock. The calves stand on the tips of their toes, and on the hind legs the toes are in most cases turned under. The condition improves so that the calves can' walk fairly well in a week or two.

d. Gestation length normal

e. Excessive amniotic fluid noted in many cases

f. Bull calves show more pronounced symptoms than heifer calves

The indications are that the heterozygotes are somewhat achondroplastic.

A proportionate type of dwarfism is known in Jersey cows (138). They tend to be smaller at birth but weights and measurements lie within normal range so dwarf animals cannot be identified by differences in general appearance early in life. At maturity they are distinctly smaller than normal cattle. It is due to a single autosomal recessive.

A recessive type of dwarfism has been reported in Angus (11). They were always distinguishable at birth. At varying ages, from birth to two or three months, they usually exhibited exceptionally compact, low-set, thick bodies, with short, wide head. They usually did not gain or fatten normally and after a few months did not show the same thickness and degree of finish as they had at an earlier age. The head also appeared relatively longer and narrower than at an earlier age. Bull and heifer dwarf calves were produced in approximately equal numbers. The condition is due to a single autosomal recessive gene.

The Hereford dwarf has probably received the most attention recently (112). Its occurrence in Herefords is quite frequent. The dwarfs were thick and blocky at birth, and most of them caused calving difficulties because of their characteristic greater width of body. This was especially noticeable when the mother of a dwarf was a heifer. Sometimes death occurred due to dystocia. Most of the dwarfs died before they were one year of age. The symptoms of dwarfism became increasingly pronounced with age, due to retarded growth. Most of them were chronic bloaters and some have died of that cause. Slightly bulging foreheads were common, but this alteration was not always extreme and might not be present. It was reported that the lateral ventricles of the brains of dwarfs contained more than the normal amount of fluid. In a two-year-old dwarf bull, spermatogenesis was present. No defect of the endocrine glands was found in the two dwarfs examined histologically.

A more comprehensive study was made of dwarfs in the Hereford, Angus, and Shorthorn breeds (78). All of the dwarfs had thick, heavy, labored breathing, as if they had some respiratory trouble. The mandible, with some malocclusion of the incisors with the dental pad, was longer than the maxilla. The dwarfs were low set, compact, blocky, short of neck, wide through the body, and had broad, short faces. The shortening of the long-bones and the bulging of the foreheads were common characteristics of these dwarfs within the Hereford, Aberdeen-Angus, and Shorthorn breeds. At two to four months of age, the dwarfs appeared to be stunted and potbellied and they were able to breathe only with difficulty. Physiological studies indicated that the dwarfs have a deficiency of the thyrotropic hormone. Later investigations (35)(135) indicate that the thyrotropic hormone is present.

Although the dwarfism occurring in the Angus, Hereford, and Shorthorn breeds is apparently due to a simple recessive gene, it has been suggested that more than one type of dwarfism is probably involved in each of these breeds (128). Also, the "cretin" characteristics of these dwarfs (51) (77) (150), may be confused with nonhereditary congenital deformities (70)(92). It also has been suggested that different phenotypic forms of dwarfism result from the interaction of the "comprest" and "conventional" dwarf genes (22)(70).

Akroteriasis Congenita (amputated)

Reported in the Swedish breed of Holstein-Friesian cattle (194)(195). In the homozygous condition it was characterized by an extremely reduced mandible and maxilla, forelegs amputated at the elbows, hind legs amputated at the hock joint, and a pronounced hydrocephalus. The calves died after full term. Among 115 calves produced by the mating of heterozygous bulls to their daughters, 102 were normal and 13 were amputated, which fits the expected ratio of 101:14 according to the hypothesis of a single recessive gene. The gene was probably imported from Germany and was widely spread in Sweden.

Short Spine

In the Oplandske mountain breed of cattle in Norway a gene for short spine when in the homozygous state caused an extremely short neck, thorax, and tail (144). The calves died after full term birth. In some, atresia ani was present. This condition was attributed to a single recessive sublethal gene.

Hydrocephalus

In Holstein hydrocephalic calves, the internal hydrocephalus was accompanied by a marked papilledema. The lateral ventricles were greatly distended so that only a thin layer of cerebral tissue remained between the cavity of the ventricles and the cranial bones. Both the humeri and the femurs of these animals showed marked malformation. The shafts of these bones were considerably shortened but larger in diameter than normal. "Asymmetry" (wryface) and "jumpy" (muscular incoordination) were found associated with hydrocephalus. The hydrocephalus appears to be due to a simple recessive. (25).

Recessive hydrocephalus has been reported in the Marche breed (62). In many cases, hydrocephalus was accompanied by malformation of the limbs. Recessive hydrocephalus also has been reported in the Hereford and Holstein-Friesian breeds (2)(105).

In a report covering the general consideration of hydrocephalus in calves, it was stated that congenital internal hydrocephalus of the newborn may well account for a considerable number of unexplained losses in calves during the first few weeks

postpartum. It is a condition which is apparently not readily recognized at birth or shortly thereafter (16).

Skull Defect

Reported in Holstein-Friesian cattle. The brain tissue protrudes from a frontal opening in the skull (165). The mode of inheritance is not known.

Nervous Disorders

There appear to be several forms of paralysis. In some cases the condition cannot be considered as a true lethal.

An abnormality characterized by incontinence of urine, partial or complete paralysis of the tail, ataxic movement of the hindquarters, and enlarged abdomen was observed in 25 young calves of the Meuse-Rhine-Yssel breed (182). The condition is due to a single recessive.

A spastic syndrome has been reported in cattle (159). It is probably a disease of the central nervous system, usually affecting older cattle. It is characterized by spastic contraction of muscles of one or both hind legs, back, and eventually the entire body. The cramps last from several seconds to a few minutes or longer and then cease suddenly, only to be repeated upon the proper stimulus. The attacks are mild for several years before a severe attack occurs. Symptoms are most evident when the animal first gets up or is startled, and are absent when the animal is recumbent. The animals appear to be less affected when on pasture, exercised moderately, and kept isolated and quiet. This characteristic has been observed in Holstein, Guernsey, Ayrshire, and crossbred Brahman and Shorthorn. It has definite familial occurrence but the exact hereditary nature has not been determined.

Congenital ataxia has been described in Jersey calves in which the symptoms are present at birth (162). The calves were well formed and appeared bright and alert, but walked like a ballet dancer and would fall down easily. It is due to a lack of development of nerve cells, axon cylinders, myelin sheaths, and algiodendroglia in the cerebellum and midbrain. Due to a simple recessive gene.

Congenital hereditary paralysis has been described in Red Danish cattle (23). Affected calves are inclined to be down with their legs fully extended. The joint and tendon reflexes are accentuated, and a tendency to clonus is noticeable. This increases tonus or spasticity and produces an apparent paralysis. Coordination of the movements of the hind legs is affected, but to a somewhat less degree. Due to a single recessive gene, homozygotes are paralytic whereas heterozygotes are phenotypically normal.

Paralyzed hindquarters have been reported in American Red Danish cattle (34). It is present at birth and the animals die at about two weeks of age. Due to a single autosomal recessive.

A muscle contracture has been described in Holstein-Friesian cattle. The calves are full term. The head is drawn up toward the back, apparently by contraction of the cervical muscles. The neck is extremely rigid. The fore and hind limbs are folded and almost wrapped around the body. They also are extremely rigid (99). Due to a simple recessive.

Paresis of the hind legs and blindness are inherited as a simple recessive in the Norwegian Red Polls (181). The calves are normal size and alive at birth. The hind legs are paralyzed, forelegs normal. Most of the calves had spasms in the neck which caused them to throw the head and neck backward. Some had acute keratitis and became blind.

The Norwegian Red Polls also have a muscle contracture caused by a simple recessive gene (181). The calves are full term. The hind legs are bent backward to a considerable degree. The forelegs are slightly bent. All legs are ankylosed. The hair coat is abnormally short and frizzled.

A prenatal tendon contracture is described in Milking Shorthorns (36). The calves are full term, but show abnormal development of the skeletal muscle. The suspensory apparatus of the front legs is in a contracted state. It is actually necessary to sever the flexon tendons to straighten the legs. Due to a simple recessive. A similar condition is reported in Gir and Indo-Brazilian Zebus.

Hereditary congenital lethal spasms are also known in Jersey cattle (73). The calves appear robust and healthy except for certain convulsive movements. They can stand but never do so voluntarily. The condition is due to a simple recessive.

In the Brown Swiss, an epileptic-type character is inherited as an autosomal dominant (8). It is characterized by a lowering of the head, tongue chewing, slight foaming at the mounth, and finally collapse in a coma.

A check list of hereditary and familial diseases of the central nervous system in domestic animals has been compiled (161). Some of the selected examples are:

Breed	Character	Inheritance
1. Brown Swiss	Epilepsy	Dominent
2. Danish Red	Congenital posterior paralysis	Recessive
3. Hereford	Ataxia, cerebellar hypoplasia	Not reported
4. Holstein-Friesian	Spastic paresis	Recessive
5. Swiss Spotted	Spastic paresis	Recessive
6. Holstein-Friesian	Spasms	Recessive
7. Jersey	Spasms	Recessive
8. Norwegian Red Poll	Paralysis, spasms of neck	Recessive
9. Simmental	Tremor of forelimbs	Not reported

Ankylosis

Characterized by ossification of the articulation of the lower jaw and shortening of the jaw. The affected animals are stillborn or die soon after birth. Reported in Norwegian Lyngdal and German cattle. Due to a simple recessive (166).

A combination muscle contracture and ankylosis is reported in the Jersey breed (170). The forelegs are usually deformed and, in addition, some of the calves have a wryneck, flexure of the spinal column, and an incompletely developed palate. It is reported in an inbred herd, but the mode of inheritance is not given.

Jaw Deformity

A deformity of the lower jaw has been reported in Milking Shorthorns (3). The jaw is only half normal length. It is due to a recessive gene. A malocclusion due to mandibular prognathism is also reported due to a dominant gene with reduced penetrance (171).

Agnathia has been reported in various degrees. In the Ayrshire, the upper jaw is 10 cm. longer than the lower jaw. The tongue is narrow and small (126). In Jersey cattle, it is characterized by a greatly shortened lower jaw that either lacks teeth or has undeveloped dentition (189). The eyes appear sunken, with an

unbroken hairless skin or membrane over the sockets. The ears are shortened and abnormal. There are large, hairless areas over the body, especially below the knees and hocks. Due to a simple recessive. Another type of agnathia in the Jersey is described as an ossification of the entire mandibular space. The lower jaw was very short and the animal could not breathe or swallow. Limited to males, this was due to a recessive sex-linked factor (52). In the New Zealand Shorthorn cattle, there is complete absence of the lower jaw (43). Due to a simple recessive. In the Milking Shorthorn, the premolar teeth are impacted in the lower mandible, which was greatly reduced in size (94). The calves are normal in size at birth but die within the first week. Due to a simple recessive.

Skin and Hair Defects

Hairless calves, stillborn and usually premature, are present in Japanese cattle (106). The skin is wrinkled and cracked like an elephant's hide. The condition, called "hypotrichosis hypothyreosa" is caused by hypofunction of the thyroid. Due to a simple recessive factor.

In the Holstein, an epithelial defect is characterized by absence of the hair on the skin below the knees and hocks, one or more undeveloped claws, deformed ears, defects in the muzzle, and virtually complete absence of mucous membrane (82)(83)(84). The condition is called "epitheliogenesis imperfecta neonatorium bovis" and is due to a simple recessive gene.

A very similar condition has been reported for Jersey cattle (155). It is also due to a simple recessive gene and it was suggested that the gene is probably common for Holstein and Jersey.

Another simple recessive gene in the Swedish Holstein-Friesian causes animals to be completely hairless (143). This condition has been designated "hypotrichosis congenita."

Holsteins also have a defect characterized by a deficiency of the front teeth and a deficiency of hair on the head and neck. As the animals become older they appear normal (26). The mode of inheritance is not known.

A condition called "streaked hairlessness" is also reported in Holstein-Friesian cattle. The affected animals are devoid of hair on various parts of the body, the hairless areas occurring in more or less consistent patterns (47). Only the females are affected. There is an abnormal sex ratio of 2 females to 1 male born. It was concluded that the condition is due to a semidominant, sex-linked, lethal gene. In the Normandy and Maine-Anjou x Charollais cattle, a congenital condition marked by hairlessness, toothlessness, deficiency of the horny tissue, and enlargement of the tongue is reported (42). It appears to be a sex-linked recessive transmitted by the female to some of her sons.

A condition has been described in Guernsey cattle in which the animals are completely hairless except for the tip of the tail and the inside of the ears (100). The teeth, hoofs, and horn buds appear normal. At maturity some normal hairs are present inside of the ears and the lower portions of the legs. Otherwise, only sparse, scraggly hairs are present. It is due to a simple autosomal recessive character.

In Ayrshire cattle, bare areas may occur on the legs, muzzle, and inside of the mouth. It is due to a simple recessive gene (99).

A condition of semihairlessness has been described in calves resulting from a purebred Polled Hereford bull used on mixed grade cows (33). The affected calves

do not grow well and appear to be very wild. They are normal in size at birth but very thin. They have a very thin coat of short, fine, curly hair at birth and in two or three weeks coarser hair begins to appear, but they never develop a normal hair coat. A very similar condition was noted in Polled Hereford cattle in California (115). The condition is due to a simple autosomal recessive.

A "karakul" type of curliness has been described in Ayrshire (49) and in Swedish cattle (109). In both cases the condition is due to an autosomal dominant gene.

Foot and Hoof Defects

Syndactylism has been reported in the Holstein-Friesian (50) and in the Hariana breed (113)(167). It is due to a simple recessive.

Polydactylism is present on the front foot of Hereford males (145). As the animals get older the feet become tender and the animals are lame. It is due to a sex-linked recessive. In the Holsteins, polydactylism is due to a dominant gene (68).

Reduced phalanges in Swedish cattle is due to a single recessive (98).

A condition has been described in Jersey cattle in which there is a lack of tone in the muscles and ligaments holding the phalanges together (142). The condition is due to a single autosomal recessive factor. Flexed pasterns also has been noted in Jersey cattle (6)(139). It is due to a single autosomal recessive gene (139). A method of measuring the metatarsal inclination in Ayrshire is described (81). The inclination is inherited.

A congenital hereditary lameness is described in Red Danish cattle (23)(146)(153a). It has become rather widespread in some areas. Due to a single recessive gene.

In Glan cattle the occurrence of warts between the hoofs is due to a dominant gene (65).

Porphyrinuria (pink tooth)

This condition has been studied in detail in a herd of Shorthorn cattle in South Africa (58)(59)(60). It is an abnormality of pigment metabolism in which young animals lose hair and develop a rough coat. Crusts and scabs may be present around the eyes and nose, and there may be a nasal discharge. The urine is red and may remain so for several months. The teeth and bones are a reddish brown color. It is due to a simple autosomal recessive.

Congenital Dropsy

Affected calves may show degrees of dropsy from slight to severe. Slight cases may pass unrecognized, while severe cases often entail loss of the dam (39). Forms of dropsy have been reported in Ayrshire and Swedish Lowland cattle (48). The condition is due to a recessive gene which may be favored in the heterozygous condition since the incidence in pedigreed herds in England is increasing rapidly (39).

Eye Cancer and Epithelioma

Skin cancers and dermatitis are usually found in cattle with white hair and a white skin. If cattle with white coat and hide eat certain plants, the hide becomes supersensitive to radiation (17).

Hereford cattle, with their white faces and unpigmented eyelids, are susceptible to eye cancers and epithelioma. The cancers develop exclusively on the conjunctiva in breeds where the conjunctiva is deficient in pigment. Cancers also develop on those parts of the eyelids which are not covered with hair and which are unpigmented. Epithelioma is probably due to the effect of solar radiation and occurs on the conjunctiva and the hairless, unpigmented rim of the eyelids (17)(61)(80)(179)(191).

Epithelioma of the eyelids may be almost entirely prevented by breeding only from those Hereford cattle which have a ring of brown hair around the eyes and pigmented eyelids. Apparently, there is a close connection between the amount of color of the hair about the eye and the amount of pigment present in the conjunctiva (17)(179).

In addition, there is an apparent tendency for susceptibility to eye cancer to be inherited (80)(179)(191).

TABLE I

Variation in Pigmentation about the Eyes of Hereford Cattle (17)

Herd	Classification According to Amount of Pigmentation			Incidence of Opthalmia, Other Affliction and Cancer		
	++	+	-	++	+	-
A	19	252	59	0	16	25
B	9	63	98	0	0	26
C	3	48	27	0	5	21
D	16	137	73	0	17	28
Mara	55	159	102	0	17	22
Total	102	659	359	0	55	122
Percentage of total	9.1	58.8	32.1	0	4.9	10.9
Percentage each class				0	8.3	34.0

\- Pigment around eye absent.
\+ Pigment around eye small, often interrupted; also, pigment or broken pigment on eyelids.
++ A large ring at least one-half inch wide around the eyes.

In a study of 400 cancer eyes, the following results were obtained (179):

a. All cancers developed on mucous membrane lining the eye, and on the cornea. In no instance did the cancer develop on the skin.

b. Lid pigmentation in cancer eyes.
   1. Complete pigmentation - 8%
      Partial pigmentation - 58%
      No pigmentation - 34%

c. Location of cancer:
   1. Cornea - 50%
      Lid - 22%
      Nictitating membrane - 17%
      Questionable - 11%

Congenital Cataract

The condition has been reported in Holstein-Friesian and Jersey cattle (38)(68)(72). The lens of the eye shows an opaque body beneath the cornea. It is due to a single autosomal recessive gene.

Strabismus

The cross-eyed condition is not always evident at birth, but does show by the time the animal is a year old (154). Due to a single autosomal recessive.

Night Blindness

The animals were affected from the time of birth (32). Described in Milking Shorthorns, the animals could see in sunlight but not after dark. There was no visible defect of the eyes. The mode of inheritance was not determined.

Muscular Hypertrophy

Reported in Herefords, Angus, Galloway, Holstein, Ayrshires, Shorthorns, Charollais, and crossbred Africander-Angus (87)(116)(185). It is due to a recessive gene, but the heterozygote may be favored in selection. The thighs of the affected animals are extremely deep and full and show a deep groove. Because of the additional size, parturition is difficult.

Udder Abnormalities

A faulty placement of the fore and rear teats in the Hereford is inherited as a simple recessive (111).

In the Guernsey, a condition has been described in which the udder is poorly shaped on the right side and only one teat present on the left side (93). Due to a simple recessive. It has also been noted that in Holstein x Galloway crosses acute mastitis appears to be present in certain cow families (186). The exact mode of inheritance was not determined.

Wrytail

Wrytail has been reported in Jersey, Guernsey, Holstein, Ayrshire, Brown Swiss, and Red Polled cattle (7)(9)(119). It is due to a simple autosomal recessive but the direction of tail set is nongenetic.

Ear Defects

"Double ears" in Brahman cattle is caused by a thin, flat piece of cartilage parallel to the main axis of the ear and projecting out of the back surface of the ear very much as the dorsal fin of a fish projects from its back (132). Due to a simple dominant gene. In Jersey and Ayrshire cattle, a notch on the lower edge of the ear similar to an identification notch is inherited as a dominant (31)(132). In Ayrshires, it was reported that 1.4 percent have wrytails, .09 percent have wry faces, and .83 percent have cropped ears (31).

Umbilical Hernia

Appeared in Holstein-Friesian male calves between the ages of 8 and 20 days. The condition persisted in varying degrees until the cattle were between 10 weeks and 7 months of age when the sac seemed to contract (183). It appears to be due to a sex-limited autosomal dominant gene.

TABLE II

Summary of the Recessive Genes Carried by Six Sires Selected at Random (141)

Sire	Epitheliogenesis Imperfecta	Recessive Achondroplasia	Proportionate Dwarfism	Congenital Cataract	Strabismus	Flexed Pasterns	Female Sterility
3000	-	+	-	-	+	+	-
300	-	-	-	+	-	-	+
300A	+	-	-	-	-	+	-
300C	-	-	+	-	-	-	+
300D	-	-	-	-	+	-	++
100B	-	-	-	-	-	-	+

- Defect did not appear in test progeny, assumed to be homozygous normal.
+ Heterozygous for specific recessive gene.
++ Homozygous for specific recessive gene.

Disease

There are reported cases of Gujarati Zebu cattle being immune to foot-and-mouth disease. The Zebu are also immune to Texas fever and anthrax and will transmit this immunity to their F_1 progeny (121). The Nellore cattle are also highly resistant to rinderpest.

There is a recognized genetic difference in the ability of animals to withstand disease or infection (69), and in some animals the resistance has been found to be monogenic (127).

Miscellaneous Inherited Factors

There are inherent differences between the progeny of different bulls in their ability to handle large quantities of feed without digestive disturbance (118).

Many animal behavior characteristics, including such items as grazing time, ruminating time, the distance an animal will walk, the number of drinks an animal will take in a day, appear to be inherited (88)(90)(91).

Genetic factors have also been recognized in the establishment of social hierarchies for animals (29)(32).

In a summary of cattle inheritance, the following characteristics were listed (68):

a. Black tongue dominant to white
b. Voice of bison and hump of bison dominant
c. Dewlap and sheath of *indicus* dominant or partially dominant to *taurus*
d. Small size of Holstein front teeth dominant

Color Inheritance

In various crosses within and between beef and dairy breeds, black is dominant to red and to white (20)(27)(66)(130)(184). Brindle in Telemark cattle is dominant to red (193). A study of West Coast cattle of Norway indicated the following (14):

a. Brown color is determined by the factor Bs, dominant to red and recessive to black. Brown color is not an allele to black.

b. Brindle is determined by the factor Br, acting on the brown factor. It has no influence on black and red.

c. Dun is determined by a factor D, dominant to black. It has no influence on red.

In crosses of white Shorthorn x Galloway it was postulated that white is caused by the absence of the extension factor E, (130).

In crosses of dairy and beef breeds, the following results were obtained (66):

a. There is a recessive dilution factor in the Guernsey breed.
b. Inguinal white is dominant.
c. Pigmented muzzle is dominant to unpigmented muzzle.
d. Pigmented tongue is dominant to unpigmented tongue.
e. Black switch is dominant to other switch colors.

The silver gray in Guzerat and Nellore cattle is due to nonpigmented tipping of the hairs (157). The tipping is inherited as recessive to nontipped condition.

Black coat color of Dutch Belted cattle is dominant to Jersey red (12). The white belt is also dominant to the solid color. The recessive white spotting of the Jersey is dominant over recessive red of the Dutch Belted. Belt and self-color are both considered dominant over absence of belt and not self-colored (122).

In a rather complete study of color inheritance in Hereford cattle, the following results were obtained (152):

a. The reduction of pigmented areas, such as line back, due to recessive factors

b. The extension of pigmented areas, such as a red neck, due to dominant factors

c. Pigment around the eye (red eye) is incompletely dominant and may be associated with red neck

d. Pigmented nose, due to dominant factors

e. Claret color is recessive to pale brown coat color

The black areas appearing in the Hereford coat appear to be inherited, but the mode of inheritance was not determined (95). The gene for Hereford pattern, Sh, is allelomorphic to self S and to recessive white spotting s. The gene Pl causes large pigmented spots on the face and causes pigmented legs (104).

Based on observed results, it has been postulated that all cattle are homozygous for the gene for black pigment, B. The extension factor E acting on B results in a color like the Angus. The gene for blackish, Bs, acting on B results in colors like the Ayrshire and Jersey (15). The gene for red, C, is present in all pigmented animals. Bs is hypostatic to E (103).

A recent study using identical twins has shown the following results (89):

a. R (red) present in all cattle except albinos. Hypostatic to other nonallelomorphic genes.

b. E (black extender) all animals are genetically black, B, but the black pigment is carried to the hairs only if the animal has E.

c. Bs (blackish) is influenced by environment. In a Bs animal, the skin is black but a strip of brownish skin runs from the lower jaw down the throat, brisket, belly, and udder. The inner surface of the ears is also brown-skinned.

d. Be (black ears) has more effect than Bs.

e. Br (brindle) can act only in the presence of Bs.

f. S represents self-color. s inhibits hair and skin pigment in certain areas.

Shorthorn Colors:

The single-factor hypothesis for roan color was advanced as the simplest explanation (158)(192).

W W = red
W w = roan
w w = white

A modification of this scheme includes an extension factor (44).

R = red
r = white
E = roan extension factor
Red = RRee, RREE, RREe
Roan = Rr EE, Rr Ee
White = rr EE, rr Ee, rr ee

Another modification includes the gene for self-color, S (168).

R = red color (incomplete dominant)
r = white
Rr = roan
S = self-color (dominant)
s = pied
Red = RR SS or RR Ss
Red-and-White = RR ss
Roan (even) = Rr SS or Rr Ss
Roan (uneven) = Rr ss
White = rrss, rr Ss, or rr ss

Incomplete experimental results with blue-gray cattle indicate that the F_2 data fit the hypothesis of either one factor of two factors involved in Shorthorn color, while backcross data do not fit either hypothesis (54). It was suggested that white acts differently with roan from the way it acts with a solid color.

Albinism:

Albinism in water buffalo, Brown Swiss, and Holstein cattle is due to a recessive gene (28)(37)(151). It is suggested that albinism is due to a lack of the enzyme tyrosinase (151).

The information on color inheritance has been summarized with appropriate symbols (101):

1. Black (B). This gene makes all pigmented hairs black. b is the absence of black. bb animals are usually red.

2. Black Spotting (Bs). Also called "blackish." This is the type of black found in Jerseys, Ayrshires, and Brown Swiss. It causes black pigmentation in certain areas. Specific modifying genes influence the degree of black. The character is also sex-influenced, being more pronounced in males.

3. Dilution. A recessive dilution factor (i) occurs in the dairy breeds Jersey and Guernsey. This is not carried by the other red breeds, such as Herefords, red Shorthorns, and Ayrshires. There is also a dominant dilution factor (D) which acts on black (B) to produce dun. The genetic explanation for dark red and yellow Herefords has not been determined.

4. Self and Recessive White Spotting. (S and s). The gene S causes an animal to be entirely pigmented. An animal that is entirely pigmented (shows no white spotting) is called "self" or "self-colored." The gene s represents white spotting. S is not completely dominant to s. There are undoubtedly many genes which influence the expression of the recessive white-spotting gene, and specific modifying genes have been postulated to account for some of this variability. The gene Lw causes a small amount of white and is incompletely dominant to its allele, lw. The gene Pl causes considerable pigmentation of the legs below the knees.

5. Roan (N). It has not been possible to establish Shorthorns that will breed true for the roan color. There are many conflicting theories to explain the mode of inheritance of roan, but the single-factor hypothesis still seems to be the best suited. According to this hypothesis: NN = red, Nn = roan, and nn = white. Part of the difficulty in studying the inheritance of this coloring has been the inability always to differentiate between a dark roan and a red. There are unquestionably many modifiers which could cause considerable variation in expression of these major genes.

6. Dominant White Spotting.

   A. Hereford Pattern: The gene for the Hereford pattern is part of an allelomorphic series:

      S^H = Hereford pattern
      S = Self (a single color, e.g., Angus)
      s = Recessive white spotting

   S^H is incompletely domant to S and completely dominant to s. The genes Lw and lw influence the amount of white that will be present.

Other modifying genes are:

Rn = Red neck. Rn is incompletely dominant to rn.
Re = Red eye (red hair around the eye). Is completely dominant to re.

Line-back: The term line-back does not mean the same to all Hereford breeders. Various definitions are used. In general, it consists of white hairs extending along the back from the end of the white crest and is apparently inherited in a quantitative nature. Since the extent or degree of white is not consistent, it is probably influenced by modifying genes, although it is likely that a major recessive gene is involved.

B. Dutch Belt (S^D). A Dutch Belt is characterized by a white belt, varying in width and regularity, around the body back of the forelegs. This is due to a gene which is part of the allelic series containing S^H and S.

S^D = Dutch Belt
S^H = Hereford Pattern
S = Self
s = Recessive White Spotting

S^D is dominant to S and s and can act with S^H.

C. Colorsided (S^C). Colorsided animals are pigmented on both sides of the body and the remainder is white. This gene is also part of the allelic series:

S^C = Colorsided
S^D = Dutch Belt
S^H = Hereford Pattern
S = Self
s = Recessive White Spotting

S^C is incompletely dominant to S and completely dominant to s.

7. Dominant White Spotting.

A. Inguinal White (In). This character is especially noticeable in some individuals of the Angus breed. The gene In causes white in the inguinal region, including the scrotum or udder.

B. Whitening (w). A recessive gene w is responsible for the "fawn" color in Jersey cattle.

C. Pigmented (Black) Skin Spotting (Ps). The dominant gene, Ps, is responsible for producing the black muzzle color and black skin spots, which may occur anywhere on the body.

Breed-color Formulas:

The possible Mendelian formulas for the color constitution of some breeds have been presented by Ibsen (101). Using the genes that have been described here, some of these formulas would be as follows:

1. Aberdeen Angus.

   BB dd II nn SS WW Inin
   inin

2. Hereford.

   bb bsbs dd II nn psps $S^{H}S^{H}$rnrn rere lwLw plpl WW
   PsPs Rnrn Rere Lwlw
   lwlw

3. Shorthorn.

   bb bsbs dd II NN psps ss LwLw RnRn PlPl WW
   Nn Psps Lwlw
   nn lwlw

4. Holstein-Friesian

   BB dd II nn psps ss LwLw plpl WW
   Psps Lwlw
   lwlw

Although the color inheritance discussed here is not complete, it is still evident that color is not a simply inherited character. The color formulas indicate that even the purebred animals are not true-breeding for color.

Color and Production

There are reported indications that dark red Hereford calves were heavier at weaning and yellow cows weaned heavier calves (163). These differences were not statistically significant.

Stockmen often argue about the relative gaining ability of light red and dark red Herefords. Some prefer the cherry reds. Others maintain that the light or "soft" reds are superior. Since all cattle used in the grazing tests are numbered and weighed individually, it has been possible to get some information on this subject. A four-year average of most cattle showed no significant difference in gains of the different color types (137).

Gains from Different Color Shades of Hereford Steers (137)

Shade of Color	Cattle Average (Number)	Four-year Average	
		Initial Weight (Pounds)	Gain Per Head (Pounds)
Soft red	390	412	357
Intermediate	1018	414	359
Dark red	388	423	354

Heritability studies with Hereford cattle indicate that approximately three pairs of additive genes determine the intensity of red color (120). There was no clear indication of dominance.

In a study of coat color and temperament (114), it was noted that brindled Durham cattle were wild while black Durhams were tame. Black Spanish cattle were also very tame.

It also has been noted that differently colored cattle in the Falkland Islands keep separate (180).

Horn Inheritance

Polled is dominant to horned but there do appear to be other factors influencing the expression of the single gene. Although some data do fit the pattern of simple monofactorial inheritance (130)(184), other data indicate a sex influence (24). In Hereford matings of horned x horned, all male calves were horned and all female calves polled. In subsequent matings of horned bulls on these polled heifers, horned male calves and polled female calves resulted. It also has been postulated that the simple dominance of the polled condition is derived from earlier sex-limited conditions which are still to be found in certain types of cattle, especially those in which there has been little or no admixture of the recognized domestic breeds (169).

A scheme whereby four genes are used to account for the polled and horned condition has been presented (187).

H = gene for horns. All cattle are assumed homozygous for this gene.
P = polled gene.
Sc = scurred gene. It is sex-limited to the extent that in both the heterozygous and homozygous condition it is epistatic to P in males, while it is epistatic only in those females that are homozygous for the gene.
Ha = "African horn." It is epistatic to P in males.

In nonpolled animals (PP), H is epistatic to Sc in both sexes and apparently is epistatic to Ha, although H and Ha may have modifying effects, making the epistacy incomplete.

Recent studies with polled Hereford cattle appear to substantiate the four gene hypothesis, although the scurred factor may be a recessive gene instead of a dominant (188).

Blood Groups of the Bovine

More than forty blood factors have been reported in the bovine erythrocytes, each of which behaves as a simple Mendelian dominant. The factors are not inherited as independent entities but appear as definite groups which in turn appear to be controlled by single genes (174).

When breeds of cattle are compared for the frequencies of the various blood groups and factors, significant differences are usually noted (148)(149).

The blood typing technique has been used for nonparentage determinations (55). In addition, the application of this technique has been described as (56)(173)(174):

a. Identification of individual animals
b. Diagnosis of monozygotic and dizygotic twins
c. Early diagnosis of freemartin
d. Measure of residual heterozygosity of inbred animals
e. Use of "marker" genes
f. Study of gene interaction
g. Selected matings for inbreeding and crossbreeding

Literature Cited

1. Anantakrishnan, C. P., A. J. Lazarus, and M. C. Rangaswamy. 1952. Observations on some Indian cattle. II. Some causes for the variation in the length of gestation. Indian J. Dairy Sci. 5:63-77. (Abs. 697). Anim. Breeding Abs. 1953. 21(2):144.

2. Anderson, W. A., and C. L. Davis. 1950. Congenital cerebellar hypoplasia in a Holstein-Friesian calf. Amer. Vet. Med. Assoc. J. 117:460-461.

3. Annett, H. E. 1939. Note on a new recessive lethal in cattle. J. Genet. 37:301-302.

4. Arrillaga, C. 1949. Dwarf cattle for the tropics. J. Hered. 40:167-168.

5. Atkeson, F. W., F. Eldridge, and H. C. Fryer. 1943. An unusual case of twinning in Jersey cattle. J. Hered. 34:81-82.

6. Atkeson, F. W., F. Eldridge, and H. L. Ibsen. 1943. Bowed pastern in Jersey cattle. J. Hered. 34:25-26.

7. Atkeson, F. W., F. Eldridge, and H. L. Ibsen. 1944. Prevalence of wrytail in cattle. J. Hered. 35:11-14.

8. Atkeson, F. W., H. L. Ibsen, and F. Eldridge. 1944. Inheritance of an epileptic type character in Brown Swiss cattle. J. Hered. 35:45-48.

9. Atkeson, F. W., and T. R. Warren. 1935. Inheritance of wrytail in Jersey cattle. J. Hered. 26:331-334.

10. Baker, M. L., C. T. Blunn, and M. M. Oloufa. 1950. Stumpy, a recessive achondroplasia in Shorthorn cattle. J. Hered. 41:243-245.

11. Baker, M. L., C. T. Blunn, and M. Plum. 1951. "Dwarfism" in Aberdeen-Angus cattle. J. Hered. 42:141-143.

12. Becker, R. B. 1933. Recessive coloration in Dutch Belted cattle. J. Hered. 24:283-287.

13. Becker, R. B., and P. T. D. Arnold. 1949. "Bulldog head" cattle; prognathism in grade Jersey strain. J. Hered. 40:282-286.

14. Berge, S. 1949. Inheritance of dun, brown, and brindle color in cattle. (Abs.) Internatl. Cong. Genet. Proc. (1948)8:535-536. Heredity 3:195-204.

15. Bogart, Ralph, and H. L. Ibsen. 1937. The relation of hair and skin pigmentation to color inheritance in cattle, with some notes on guinea-pig hair pigmentation. J. Genet. 35:31-59.

16. Bone, J. F. 1953. Hydrocephalus in calves. No. Amer. Vet. 34:25-28.

17. Bonsma, J. C. 1949. Breeding cattle for increased adaptability to tropical and subtropical environments. J. Agr. Sci. 39:204-221.

18. Boyd, W. L. 1946. A clinical study of "white heifer disease." Cornell Vet. 34:337-345.

19. Brandt, G. W. 1941. Achondroplasia in calves. J. Hered. 32:183-186.

20. Campbell, M. H. 1933. Inheritance of black and red coat colors in cattle. Genetics 9:419-441.

21. Carmichael, J. 1933. "Bulldog" calf in African cattle. Nature 131:878.

22. Chambers, D., J. A. Whatley, and D. F. Stephens. 1954. The inheritance of dwarfism in a comprest Hereford herd. (Abs.) J. Anim. Sci. 13:956-957.

23. Christensen, E., and N. O. Christensen. 1952. Congenital hereditary paralysis in calves. A clinical and pathological-anatomical study. Nord. Vetmed. 4:861-874.

24. Churchill, O. O. 1927. Sex and horns in cattle. J. Hered. 18:279-281.

25. Cole, C. L., and L. A. Moore. 1942. Hydrocephalus, a lethal in cattle. J. Agr. Res. 65:483-491.

26. Cole, L. J. 1919. A defect of hair and teeth in cattle probably hereditary. J. Hered. 10:303-307.

27. Cole, L. J., and S. V. H. Jones. 1920. The occurrence of red calves in black breeds of cattle. Wis. Agr. Expt. Sta. B. 313.

28. Cole, L. J., E. E. VanLone, and I. Johansson. 1924. Albinotic dilution of color in cattle. J. Hered. 25:145-156.

29. Collias, N. E. 1944. Aggressive behavior among vertebrate animals. Physiol. Zool. 17:83-123.

30. Collias, N. E. 1950. Social life and the individual among vertebrate animals. N. Y. Acad. Sci. Ann. 51:1074-1092.

31. Conklin, C. T. 1949. Nature's quirks in the dairy herd; wrytails, wry faces, and cropped ears becoming more widely distributed. Ayrshire Digest 35:886-887.

32. Craft, W. A. 1927. Night blindness in cattle. J. Hered. 18:215-216.

33. Craft, W. A., and W. L. Blizzard. 1934. The inheritance of semihairlessness in cattle. J. Hered. 25:385-390.

34. Cranek, L. J., and N. P. Ralston. 1953. Paralyzed hindquarters; a hereditary defect in American Red Danish cattle. (Abs.) J. Anim. Sci. 12:892-893.

35. Crenshaw, W. W., and C. W. Turner. 1954. Estimating the thyrotropin and thyroxine secretion rates of cattle. (Abs.) J. Anim. Sci. 13:1017.

36. Dale, D. G., and J. E. Moxley. 1952. Prenatal tendon contracture in a herd of Milking Shorthorns. Canad. J. Compar. Med. 16:399-404.

37. Detlefson, J. A. 1920. A herd of albino cattle. J. Hered. 11:378-389.

38. Detlefson, J. A., and W. W. Yapp. 1920. The inheritance of congenital cataract in cattle. Amer. Nat. 54:277-280.

39. Donald, H. P., D. W. Deas, and A. L. Wilson. 1952. Genetical analysis of the incidence of dropsical calves in herds of Ayrshire cattle. Brit. Vet. J. 108:227-245.

40. Donald, H. P., and J. L. Hancock. 1953. Evidence of gene-controlled sterility in bulls. J. Agr. Sci. 43:178-181.

41. Downs, W. G., Jr. 1928. An American "Dexter monster." Anat. Rec. 37:365-372.

42. Drieux, H., M. Priouzeau, G. Thiery, and M. L. Priouzeau. 1950. (Congenital hairlessness, toothlessness, deficiency of the horny tissue, and enlarged tongue in the calf.) Rec. de Med. Vet. 126:385-399. (Abs. 1209. Anim. Breeding Abs. 1953. 21(3):252.)

43. Dry, F. W. 1938. Genetics and livestock production. New Zeal. Dept. Sci. & Indus. Res. B. 64.

44. Duck, R. W. 1923. Colors of Shorthorn cattle. J. Hered. 14:65-75.

45. Dutt, M. K., and P. Bhattacharya. 1952. Chromosomes of the Indian water buffalo. Nature 170:1129.

46. Eaton, O. N. 1937. A summary of lethal characters in animal and man. J. Hered. 28:320-326.

47. Eldridge, F. E., and F. W. Atkeson. 1953. Streaked hairlessness in Holstein-Friesian cattle. J. Hered. 44:265-271.

48. Eldridge, F. E., and F. W. Atkeson. 1953. Occurrence of hereditary edema in Ayrshires. (Abs.) J. Dairy Sci. 36:598.

49. Eldridge, F. E., F. W. Atkeson, and H. L. Ibsen. 1949. Inheritance of a karakul-type curliness in the hair of Ayrshire cattle. J. Hered. 40:205-214.

50. Eldridge, F. E., W. H. Smith, and W. M. McLeod. 1951. Syndactylism in Holstein-Friesian cattle. J. Hered. 42:241-250.

51. Elings, J. T. 1953. Dwarfism in beef cattle. Oreg. State Col. Ext. B. 743.

52. Ely, F., F. E. Hull, and H. B. Morrison. 1939. Agnathia, a new bovine lethal. J. Hered. 30:105-108.

53. Eriksson, I. K. 1946. Hereditary hypoplasia in cattle. J. Hered. 37:38.

54. Evvard, John M., P. S. Shearer, E. W. Lindstrom, and A. D. B. Smith. 1930. The inheritance of color and horns in blue-gray cattle. Iowa Agr. Expt. Sta. Res. B. 133.

55. Ferguson, L. C. 1947. The blood groups of cattle. Amer. Vet. Med. Assoc. J. 111:466-469.

56. Ferguson, L. C. 1951. Some applications of blood typing of cattle. Vet. Sci. News 5(3):7-13.

57. Fincher, M. G., P. Olafson, and J. Ferguson. 1942. Sterility in bulls. Cornell Vet. 32:407-423.

58. Fourie, P. J. J. 1936. The occurrence of congenital porphyrinuria (pink tooth) in cattle in South Africa. Onderstepoort J. Vet. Sci. and Anim. Indus. 7:535-565.

59. Fourie, P. J. J. 1939. Bovine congenital porphyrinuria (pink tooth) inherited as a recessive character. Onderstepoort J. Vet. Sci. and Anim. Indus. 13:383-398.

60. Fourie, P. J. J., and C. Rimington. 1937. Living animal cases of congenital porphyrinuria. Nature 140:68.

61. Frank, E. R. 1943. Neoplasms of the bovine eye. Amer. Vet. Med. Assoc. J. 102:200-203.

62. Giannotti, D. 1952. (Cases of congenital hydrocephalus in calves.) Mem. Soc. Tosc. Sci. Nat., B. 59. (Abs. 915. Anim. Breeding Abs. 1954. 22(3):207.)

63. Gilman, J. P. W., and E. W. Stringham. 1953. Hereditary umbilical hernia in Holstein cattle. J. Hered. 44:113-116.

64. Gilmore, L. O. 1949. The inheritance of functional causes of reproductive inefficiency: A review. J. Dairy Sci. 32:71-91.

65. Gottwald, W. 1954. (Warts between the hooves, an inherited defect in cattle.) Vet. Med. Diss. Freie Univ., Berlin. (Abs. 517. Anim. Breeding Abs. 22(2):118.)

66. Gowen, J. W. 1918. Inheritance studies of certain color and horn characteristics in first generation crosses of dairy and beef breeds. Maine Agr. Expt. Sta. B. 272.

67. Gowen, J. W. 1920. Inheritance in crosses of dairy and beef breeds. J. Hered. 11:300-316.

68. Gowen, J. W. 1927. A resume of cattle inheritance. Bibliog. Genet. 3:87-140.

69. Gowen, J. W. 1948. Inheritance of immunity in animals. Ann. Rev. Microbiol. 2:215-254.

70. Gregory, P. W. 1954. An analysis of wry calves in California beef herds. (Abs.) J. Anim. Sci. 13:957-958.

71. Gregory, P. W., S. W. Mead, and W. M. Regan. 1942. A new type of recessive achondroplasia in cattle. J. Hered. 33:317.

72. Gregory, P. W., S. W. Mead, and W. M. Regan. 1943. A congenital hereditary eye defect in cattle. J. Hered. 34:125-128.

73. Gregory, P. W., S. W. Mead, and W. M. Regan. 1944. Hereditary congenital lethal spasms in Jersey cattle. J. Hered. 35:195-200.

74. Gregory, P. W., S. W. Mead, and W. M. Regan. 1951. A genetic analysis of prolonged gestation in cattle. Portugaliae Acta Biol. Ser. A 1949/51: 861-882.

75. Gregory, P. W., S. W. Mead, W. M. Regan, and W. C. Rollins. 1951. Further studies concerning sex-limited genetic infertility in cattle. J. Dairy Sci. 34:1047-1055.

76. Gregory, P. W., W. M. Regan, and S. W. Mead. 1945. Evidence of genes for female sterility in dairy cows. Genetics 30:506-517.

77. Gregory, P. W., W. C. Rollins, and F. D. Carroll. 1952. Heterozygous expression of the dwarf gene in beef cattle. Sowest. Vet. 5:345-349.

78. Gregory, P. W., W. C. Rollins, P. S. Pattengale, and F. D. Carroll. 1951. A phenotypic expression of homozygous dwarfism in beef cattle. J. Anim. Sci. 10:922-933.

79. Gregory, P. W., C. B. Roubicek, F. D. Carroll, P. O. Stratton, and N. W. Hilston. 1953. Inheritance of bovine dwarfism and the detection of heterozygotes. Hilgardia 22:407-450.

80. Guilbert, H. R., A. Wahid, K. A. Wagnon, and P. W. Gregory. 1948. Observations on pigmentation of eyelids of Hereford cattle in relation to occurrence of ocular epitheliomas. J. Anim. Sci. 7:426-429.

81. Habel, R. E. 1948. On the inheritance of metatarsal inclination in Ayrshire cattle. Amer. J. Vet. Res. 9:131-139.

82. Hadley, F. B. 1927. Inheritance of epithelial defects in cattle. Amer. Soc. Anim. Prod. Proc. 1927:41-44.

83. Hadley, F. B. 1927. Congenital epithelial defects of calves. J. Hered. 18:487.

84. Hadley, F. B., and L. J. Cole. 1928. Inherited epithelial defects in cattle. Wis. Agr. Expt. Sta. Res. B. 86.

85. Hadley, F. B., and B. L. Warwick. 1927. Inherited defects of livestock. Amer. Vet. Med. Assoc. J. 70:492-504.

86. Hallgren, W. 1951. (Abnormally long pregnancy in the cow.) Nord. Vetmed. 3:1043-1060. (Abs. 188. Anim. Breeding Abs. 1953.21(1):44.)

87. Hammond, J. 1935. The inheritance of productivity in farm animals. Empire J. Expt. Agr. 3:1-12.

88. Hancock, J. 1950. Studies in monozygotic cattle twins. IV. Uniformity trials: grazing behavior. New Zeal. J. Sci. & Tech. A, Agr. Res. Sect. 32(4):22-59.

89. Hancock, J. 1952. Identical twins and color inheritance. Endeavour 11:78-86.

90. Hancock, J. 1953. Grazing behavior of cattle. Commonwealth Bur. Anim. Breeding & Genet. Anim. Breeding Abs. 21(1):1-13.

91. Hancock, J. 1954. Studies of grazing behavior in relation to grassland management. I. Variations in grazing habits of dairy cattle. J. Agr. Sci. 44:420-433.

92. Hart, G. H., H. R. Guilbert, K. A. Wagnon, and H. Goss. 1947. "Acorn calves," a nonhereditary congenital deformity due to maternal nutritional deficiency. Calif. Agr. Expt. Sta. B. 699.

93. Heizer, E. E. 1932. An inherited udder abnormality in cattle. J. Hered. 23:111-114.

94. Heizer, E. E., and M. C. Hervey. 1937. Impacted molars, a new lethal in cattle. J. Hered. 28:123-127.

95. Horlacher, W. R. 1928. Exceptional color inheritance in Hereford cattle. J. Hered. 19:10.

96. Hutt, F. B. 1934. A hereditary lethal muscle contracture in cattle. J. Hered. 25:41-46.

97. Hutt, F. B. 1934. Inherited lethal characters in domestic animals. Cornell Vet. 24:1-25.

98. Hutt, F. B. 1946. Some hereditary abnormalities of domestic animals. Cornell Vet. 36:180-194.

99. Hutt, F. B., and J. N. Frost. 1948. Hereditary epithelial defects in Ayrshire cattle. J. Hered. 39:131.

100. Hutt, F. B., and L. Z. Saunders. 1953. Viable genetic hypotrichosis in Guernsey cattle. J. Hered. 44:97-103.

101. Ibsen, H. L. 1933. Cattle inheritance. I. Color. Genetics 18:441-480.

102. Ibsen, H. L. 1946. Why is the Aberdeen-Angus breed homozygous for polled? J. Anim. Sci. 5:391.

103. Ibsen, H. L 1949. The inheritance of the allelomorphs and modifiers of white spotting in cattle. (Abs.) Internatl. Cong. Genet. Proc. (1948) 8:601-602.

104. Ibsen, H. L., and A. D. Weber. 1933. The genetics of the Hereford pattern. Amer. Soc. Anim. Prod. Proc. 1933:291-294.

105. Innes, J. R. M., S. Dorothy, and A. J. Wildson. 1940. Familial cerebellar hypoplasia and degeneration in Hereford calves. J. Path. & Bact. 50:455-461.

106. Ishihara, M. 1950. Studies on the undesirable recessive genes in Japanese breed of cattle. (English summary.) Chiba-shi Zootech. Expt. Sta. Res. B. 58.

107. Jasper, D. E. 1950. Prolonged gestation in the bovine. Cornell Vet. 40:165-172.

108. Jeffreys, M. D. W. 1953. Bos brachyceros or dwarf cattle. Vet. Rec. 65:393-396.

109. Johansson, I. 1942. Reduced phalanges and curly coat. Hereditas 28:278-287.

110. Johansson, I. 1953. A new type of achondroplasia in cattle. Hereditas 39:75-87.

111. Johnson, L. E. 1945. Fused teats. A hereditary defect in beef cattle. J. Hered. 36:317-320.

112. Johnson, L. E., G. S. Harshfield, and W. McCone. 1950. Dwarfism, an hereditary defect in beef cattle. J. Hered. 41:177-181.

113. Joshi, N. R., and R. W. Phillips. 1953. Zebu cattle of India and Pakistan. Food & Agr. Organ. United Nations. FAO Agr. Studies 19.

114. Keeler, C. E. 1947. Coat color, physique, and temperament. J. Hered. 38:271-278.

115. Kidwell, J. F., and H. R. Guilbert. 1950. A recurrence of the semihairless gene in cattle. J. Hered. 41:190-192.

116. Kidwell, J. F., E. H. Vernon, R. M. Crown, and C. B. Singletary. 1952. Muscular hypertrophy in cattle. J. Hered. 43:62-68.

117. Kidwell, J. F., L. Walker, and J. A. McCormick. 1954. Hereditary female sterility in Holstein-Friesian cattle. J. Hered. 45:142,145.

118. Knapp, B., Jr., A. L. Baker, and R. W. Phillips. 1943. Variations in the occurrence of bloat in the steer progeny of beef bulls. J. Anim. Sci. 2:221-225.

119. Knapp, B., Jr., M. W. Emmel, and W. F. Ward. 1936. The inheritance of screw tail in cattle. J. Hered. 27:269-271.

120. Koger, Marvin, and J. D. Mankin. 1952. Heritability of intensity of red color in Hereford cattle. J. Hered. 43:15-17.

121. Kozelka, A. W. 1929. The inheritance of natural immunity among animals. J. Hered. 20:519-530.

122. Kuiper, K. 1921. Color inheritance in cattle. J. Hered. 12:102-109.

123. Lagerlof, N. 1951. Hereditary forms of sterility in Swedish cattle breeds. Fertility & Sterility 2:230-239.

124. Lagerlof, N., and H. Boyd. 1953. Ovarian hypoplasia and other abnormal conditions in the sexual organs of cattle of the Swedish Highland breed: Results of post-mortem examination of over 6,000 cows. Cornell Vet. 43:64-79.

125. Lagerlof, N., and I. Settergren. 1953. Results of 17 years' control of hereditary ovarian hypoplasia in cattle of the Swedish Highland breed. Cornell Vet. 43:52-64.

126. Lalonde,L. M. 1940. A new type of bovine agnathia. J. Hered. 31:80-81.

127. Lambert, W. V. 1933. The evidence for inheritance of resistance to bacterial diseases in animals. Q. Rev. Biol. 8:331-337.

128. Lindley, C. E. 1951. Observations on midgets in beef cattle. J. Hered. 42:273-275.

129. Ljutikov, K. M. 1935. Breeding within the family of the champion cow "Zolotaja." Anim. Breeding Abs. 3:369.

130. Lloyd-Jones, O., and J. M. Evvard. 1916. Inheritance of color and horns in blue-gray cattle. Iowa Agr. Expt. Sta. Res. B. 30.

131. Lucas, K., F. X. Gassner, H. H. Stonaker, and S. S. Wheeler. 1950. Relationship of thyroid, adrenal, and pituitary characteristics to body development in small and conventional type of fat Hereford steers. Western Section, Amer. Soc. Anim. Prod. Proc. 1:73-78.

132. Lush, J. L. 1930. "Double ears" in Brahman cattle. J. Hered. 15:93-96.

133. Lush, J. L. 1930. "Duck-legged" cattle on Texas ranges. J. Hered. 21:85-90.

134. Makino, S. 1951. An atlas of the chromosome numbers in animals. Ed. 2. Ames, Iowa State Col. Press. 290 pp.

135. Marlowe, T. J., and D. Chambers. 1954. Some endocrine aspects of dwarfism in beef cattle. (Abs.) J. Anim. Sci. 13:961.

136. Matthey, R. 1951. The chromosomes of the vertebrates. Advn. Genet. 4:159-180.

137. McIlvain, E. H., A. L. Baker, W. R. Kneebone, W. F. Lagrone, and E. A. Tucker. 1954. Eighteen-year summary of range improvement studies, 1937-1954. Agr. Res. Ser. U. S. Southern Great Plains Field Station, Woodward, Oklahoma.

138. Mead, S. W., P. W. Gregory, and W. M. Regan. 1942. Proportionate dwarfism in Jersey cows. J. Hered. 33:411-416.

139. Mead, S. W., P. W. Gregory, and W. M. Regan. 1943. Hereditary congenital flexed pasterns in Jersey cattle. J. Hered. 34:367-372.

140. Mead, S. W., P. W. Gregory, and W. M. Regan. 1946. A recurrent mutation of dominant achondroplasia in cattle. J. Hered. 37:183-188.

141. Mead, S. W., P. W. Gregory, and W. M. Regan. 1946. Deleterious recessive genes in dairy bulls selected at random. Genetics 31:574-588.

142. Mead, S. W., P. W. Gregory, and W. M. Regan. 1949. An hereditary digital anomaly of cattle. J. Hered. 40:151-155.

143. Mohr, O. L., and C. Wriedt. 1928. Hairless, a new recessive lethal in cattle. J. Genet. 19:315-336.

144. Mohr, O. L., and C. Wriedt. 1930. Short spine, a new recessive lethal in cattle. J. Genet. 22:278-298.

145. Morrill, E. L. 1945. A new sex-linked defect in cattle. J. Hered. 36: 81-82.

146. Nielsen, J. 1942. Investigations of hereditary lameness in the Red Danish breed. (Discuss. 100.) Nord. Jordbr. Forsk. 24:97-100. (Anim. Breeding Abs. 1943. 11:160.)

147. Oguma, K., and S. Kakino. 1932. A revised check list of the chromosome number in Vertebrata. J. Genet. 26:239-254.

148. Owen, R. D., C. J. Stormont, and M. R. Irwin. 1944. Differences in frequency of cellular antigens in two breeds of dairy cattle. J. Anim. Sci. 3:315-321.

149. Owen, R. D., C. J. Stormont, and M. R. Irwin. 1944. Incidence of inherited cellular antigens in two breeds of dairy cattle. (Abs.) J. Anim. Sci. 3:434.

150. Pahnish, O. F., E. B. Stanley, and C. E. Safley. 1952. A study of homozygous dwarfism in beef cattle. Western Section, Amer. Soc. Anim. Prod. Proc. 3.

151. Peterson, W. E., L. O. Gilmore, J. B. Fitch, and L. M. Winters. 1944. Albinism in cattle. J. Hered. 35:135-144.

152. Pitt, F. 1920. The inheritance of color and markings in pedigree Hereford cattle. J. Genet. 9:281-304.

153. Punnett, R. C. 1936. The experiments of T. H. Richey concerning the production of monsters in cattle. J. Genet. 32:62-72.

153a. Rasmussen, J. K. (with reply by J. Nielsen). 1943. Hereditary lameness in the Red Danish breed. Nord. Jordbr. Forsk. 314:144-147. (Anim. Breeding Abs. 1946. 14:224.)

154. Regan, W. M., P. W. Gregory, and S. W. Mead. 1944. Hereditary strabismus in Jersey cattle. J. Hered. 35:233-235.

155. Regan, W. M., S. W. Mead, and P. W. Gregory. 1935. An inherited skin defect in Jersey cattle. J. Hered. 26:357-362.

156. Rendel, J. M. 1952. White heifer disease in a herd of Shorthorns. J. Genet. 51:89-94.

157. Rhoad, A. O. 1936. The silver gray color in Indian cattle. J. Hered. 27:113-119.

158. Roberts, E. Color inheritance in Shorthorn cattle. J. Hered. 28:167-168.

159. Roberts, S. J. 1953. A spastic syndrome in cattle. Cornell Vet. 43: 380-388.

160. Roubicek, C. B. 1951. Inheritance in cattle and sheep. Wyo. Agr. Expt. Sta. C. 45.

161. Saunders, L. Z. 1952. A check list of hereditary and familial diseases of the central nervous system in domestic animals. Cornell Vet. 42: 592-600.

162. Saunders, L. Z., J. D. Sweet, S. M. Martin, F. H. Fox, and M. G. Fincher. 1952. Hereditary congenital ataxia in Jersey calves. Cornell Vet. 42: 559-591.

163. Sawyer, W. A., Ralph Bogart, and M. M. Oloufa. 1948. Weaning weight of calves as related to age of dam, sex, and color. (Abs.) J. Anim. Sci. 7:514-515.

164. Seligman, C. G. 1904. Cretinism in calves. J. Path. & Bact. 9:311-322.

165. Shaw, A. O. 1938. A skull defect in cattle. J. Hered. 29:319-320.

166. Shrode, R. R., and J. L. Lush. 1947. The genetics of cattle. Advn. Genet. I:209-261. Academic Press, Inc., New York.

167. Singh, S., and P. Bhattacharya. 1949. Inheritance of syndactylism in Hariana breed of cattle. Indian J. Vet. Sci. & Anim. Husb. 19:153-159. (Abs. 1368. Anim. Breeding Abs. 1950. 18(4):395.)

168. Smith, A. D. B. 1925. Shorthorn colors. J. Hered. 16:73-84.

169. Smith, A. D. B. 1927. The inheritance of horns in cattle: some further data. J. Genet. 18:365-374.

170. Spillman, A. A., O. J. Hill, and E. C. McCulloch. 1944. Congenital muscular contracture and ankylosis in Jersey cattle. (Abs.) J. Dairy Sci. 27:655.

171. Stiles, K., and J. E. Luke. 1953. The inheritance of malocclusion due to mandibular prognathism.

172. Stonaker, H. H., and R. C. Tom. 1944. "Compact" Shorthorns. J. Hered. 35:247-250.

173. Stormont, C. J. 1950. Blood groups of the bovine. Report prepared for Dr. I. J. Cunningham, Superintendent, Animal Research Station, Wallaceville, New Zealand.

174. Stormont, C. J. 1951. The F-V and Z systems of bovine blood groups. Genetics 37:39-48.

175. Stormont, C. J. 1951. The increasing importance of blood groups in livestock production. Calif. Vet. 5:20-22.

176. Stormont, C. J. 1954. Research with cattle twins. Statistics and Mathematics in Biology, pp. 407-426. Ames, Iowa. State Col. Press.

177. Stormont, C. J., and M. R. Irwin. 1948. On the differentiation of fraternal and identical twins in cattle. (Abs.) J. Anim. Sci. 7:516.

178. Sturrarrer, T. C. 1943. Bulldog and hairless calves. J. Hered. 34: 175-178.

179. The University of Texas M. D. Anderson Hospital and Tumor Institute. 1951. Conference on bovine cancer eye. Highland Hereford Breeders' Assoc., Marfa, Texas.

180. Tinbergen, N. 1953. Social behavior in animals. John Wiley & Sons, Inc. 150 pp.

181. Tuff, P. 1948. Two new lethal factors in cattle. Skand. Vet.-Tidskrift 38:379-395.

182. Van der Plank, G. M., and H. Hoiting. 1954. (Hereditary defect in cattle. Preliminary report.) Tijdschr. Diergeneesk. 79:149-150. (Abs. 930. Anim. Breeding Abs. 22(3):209.)

183. Warren, T. R., and F. W. Atkeson. 1931. Inheritance of hernia in a family of Holstein-Friesian cattle. J. Hered. 22:345-352.

184. Watson, J. A. S. 1921. A Mendelian experiment with Aberdeen-Angus and West Highland cattle. J. Genet. 11:59-67.

185. Weber, A. D., and H. L. Ibsen. 1934. The occurrence of the double-muscled character in purebred beef cattle. Amer. Soc. Anim. Prod. Proc. 1934:228-232.

186. White, W. T., and H. L. Ibsen. 1934. Inherited susceptibility to acute mastitis in cattle. J. Hered. 25:489-590.

187. White, W. T., and H. L. Ibsen. 1936. Horn inheritance in Galloway-Holstein cattle crosses. J. Genet. 32:33-49.

188. Williams, H. D., and T. Williams. 1952. The inheritance of horns and their modification in polled Hereford cattle. J. Hered. 43:267-272.

189. Wipprecht, C., and W. R. Horlacher. 1935. A lethal gene in Jersey cattle. J. Hered. 26:363-368.

190. Woodward, R. R., R. T. Clark, and J. N. Cummings. 1947. Studies on large and small type Hereford cattle. Mont. Agr. Expt. Sta. B. 401.

191. Woodward, R. R., and B. Knapp, Jr. 1950. The hereditary aspects of eye cancer in Hereford cattle. J. Anim. Sci. 9:578-581.

192. Wright, S. 1917. Color inheritance in mammals. VI. Cattle. J. Hered. 8:521-527.

193. Wriedt, C. 1919. The brindle color in cattle in relation to red. J. Genet. 9:83-84.

194. Wriedt, C. 1930. Heredity in livestock. MacMillan Company, New York. pp. 176.

195. Wriedt, C., and O. L. Mohr. 1928. Amputated, a recessive lethal in cattle. J. Genet. 20:187-216.